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WASHINGTON, D. C.

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CARDIAC FAILURE IN BRONCHIOLITIS

Case Review

George J. Cohen, M.D.

Cardiac failure as a complication of bronchiolitis is seldom mentioned and rarely elaborated on in standard pediatric texts. Nelson[11] merely mentions failure as a complication and possible cause of death, and Adams[2] in Brennemann, makes no comment at all in this regard. Because apparent cardiac failure complicating bronchiolitis has been seen at this hospital on several occasions, it was felt worthwhile to study both our own cases of bronchiolitis and the current literature.

The study here included a review of 319 cases of bronchiolitis admitted to this hospital from January 1950 through April 1955. The diagnosis of bronchiolitis was based on the following clinical picture: the child, usually under two years of age, had a rapid onset of dyspnea following an upper respiratory infection, and on examination showed tachypnea, intercostal and/or subcostal retraction, decreased air exchange, hyper-resonance of the chest, and fine, tight, expiratory wheezes. In addition there was sometimes evidence of infection in other parts of the respiratory tree, from rhinitis to bronchopneumonia. X-rays frequently showed evidence of obstructive emphysema, but sometimes nothing more than increased broncho-vascular markings. It was felt that the white blood count and differential count varied too much to be of diagnostic aid, a feeling concurred in by Stiller[3] in his study. Throat cultures usually showed normal flora or mixed pathogens. Bronchiolitis was therefore considered primarily a clinical diagnosis.

The diagnosis of congestive failure in an infant is frequently a difficult one to make with absolute certainty. It is well known that infants often have no evidence of dyspnea or pulmonary congestion with failure. Likewise, it may be difficult to detect cardiomegaly or peripheral edema. The most consistent findings are increased pulse rate and increased liver size, and these were used as criteria for the presence of failure in this series. When present, cardiomegaly is consistent with failure. Ideally, determinations of venous pressure and circulation times should be significant data in the diagnosis of cardiac decompensation, but to my knowledge no standard values have been established in infants under two years, the age range in which bronchiolitis is encountered.

METHOD AND RESULTS

Charts of all patients with a diagnosis of bronchiolitis admitted to this hospital between January, 1950 and April, 1955 were reviewed. In each

CHART 1

General Data on Patients with Bronchiolitis and Congestive Heart Failure

Age	Under 1 year	6
	Over 1 year	2
Sex	Male	4
	Female	4
Temperature	Less than 101	6
	More than 101	2
Respirations	Less than 60	2
	More than 60	6
White blood count	Less than 10,000	3
	More than 10,000	5

case the diagnosis was considered tenable on the basis of the previously mentioned criteria. Of the total 319 cases, only 8 were considered by their observers to be in cardiac failure, and all of these fit the definition of congestive heart failure noted above. No case with associated pre-existing heart disease was considered in this study.

Chart 1 shows the general statistics referable to the 8 patients in heart failure. Although no accurate tabulations were made from the data on the patients with bronchiolitis but not in failure, it was my impression that equivalent figures would have been attained. Similar statistics also appear in the reports by other authors. Of the patients who had not been treated as if in failure, none had shown any evidence of failure ac-

CHA

Data Relating to Cardiac Decom

Patient	Time from Onset of Bronchiolitis to Onset of Failure	Increase in Pulse	Increase in Liver Size	Heart Size by X-ray
1	2 days	180 on admission	1 fb to 2½ fb	normal (dilated rt ventricle at autopsy)
2	1 day	140 to 200	0 to 4 cm	slightly large
3	2 days	120 to 150	0 to 5 cm	normal
4	less than 24 hours	180 on admission	2 fb on admission	slightly large
5	1 day	140 to 160	1 fb to 2 fb	slightly large
6	3 days	130 to 200	0 to 2 fb	normal
7	4 days	180 on admission	1 cm to 3 cm	slightly large
8	2 days	200 on admission	3 fb on admission	normal

fb refers to fingerbreadth of the recording examiner.

cording to the data available on their charts. Four patients had enlarged livers, but no specific mention of pulse rate was made, nor had there been any follow-up on liver size or pulse. Therefore, these patients were not included in the "failure" series.

In Chart 2 are data from the failure cases with specific reference to the findings related to cardiac decompensation. In each case, the pulse rose while the patient was in the hospital, or was inordinately high on admission. Likewise, the liver edge became significantly palpable during the hospital course, or was unusually large on admission. Except in the case of the patient who died, there was relatively rapid improvement in the signs of failure following digitalization. Not only were hepatomegaly and marked tachycardia noted in all 8 children diagnosed as being in heart failure, but they were present in none of the other cases of bronchiolitis. In 4 of these patients the transverse diameter of the heart was more than 50 per cent of the transverse diameter of the chest on P-A X-ray. This is a notably unreliable index of cardiomegaly in infants, but it is suggestive. Two of these 4 infants had normal cardiac contours on fluoroscopy which was done after their recovery from failure. Only 2 patients had venous pressure determinations attempted; one was definitely elevated at 300 mm. H_2O. The other pressure was 120 mm. H_2O which the cardiac consultant felt was somewhat high for a 6 week old infant.

In considering possible sources of error in the diagnosis of cardiac failure, one could reasonably discount a correlation of rapid pulse with fever be-

RT 2

pensation in Bronchiolitis Patients

ECG Findings	Venous Pressure	Digitalis Preparation	Response to Digitalis
incomplete BBB	300 mm water	cedilanid i-v	died
rt axis	not done	cedilanid i-v	12 hrs: P down; liver up
not done	not done	cedilanid i-v	12 hrs: P down; liver up
normal ECG	120 mm water	tincture, oral	2 days: P down; liver up
1st degree A-V block	not done	digitoxin i-m	2 days: P down; liver ?
not done	not done	cedilanid i-v	1 day: P down; liver ?
low T waves chest leads	not done	cedilanid i-v; digalen p-o (thiomerin)	2 days: P down; liver up
not done	not done	digalen i-m	36 hrs: P down; liver up

cause in only 2 cases was the temperature over 101. Furthermore, the sudden rise in pulse rate had no relation to the respiratory status. Likewise, it was difficult to consider the liver enlargement as due to diaphragmatic depression because in no case was there mention of increased emphysema or dyspnea, or decreased air exchange *at the time the sudden liver enlargement was noted.*

Electrocardiograms did not help to make the diagnosis of congestive heart failure in 5 patients in this study who had tracings made; only 2 had definite evidence of abnormality (incomplete bundle branch block; first degree A-V block), and 1 had suggestive evidence of myocardial involvement on the basis of low flat T waves in the chest leads. Unfortunately, no subsequent tracings were available on any of the patients.

In general, the interval between onset of the bronchiolitis and onset of failure was about 2 days. In at least 2 cases (2 and 3) the respiratory status had improved slightly (based on presence of greater air exchange and looser rales) at the time failure ensued.

DISCUSSION

The *etiology* of bronchiolitis is still uncertain, and it would seem that various agents, both bacterial and viral, may be responsible, either singly or in combination. Henderson and Rosenzweig[4] found mixed flora in the majority of throat cultures from their patients with bronchiolitis. A virus, probably influenza, was considered causative by Hubble and Osborn[5], Benvard[6], and Nelson[1]. *Hemophilus influenzae* has been suspected by Adams[2], Nelson and Wood and co-workers[7], who performed an extensive bacteriologic study. However, Wood's work was undertaken with the purpose of proving *H. influenzae* as causative; other organisms cultured from tracheal secretions were not studied so seriously. Both Benvard and Pratt[8] were impressed with the epidemic nature of the illness. Our patients exhibited a wide variety of organisms, both pathogenic and benign, on throat culture.

As to *pathology*, there is rather general agreement that the pulmonary bronchioles are plugged with mucus, pus, and debris, with scattered areas of emphysema, atelectasis, edema, and congestion in the alveoli. The cardiac pathology noted by Henderson and Rosenzweig on 7 of their patients who died in heart failure, showed edema of the myocardium in 4 and nuclear degeneration in the myocardial fibers of the other 3. Pratt and Benvard both mention the occurrence of edema and increased fat droplets in the myocardium. Van Creveld et al.[9] feel that acute interstitial myocarditis with cellular infiltration may occur in almost any infectious disease. In our patient who died there were no demonstrable histologic changes in the heart, but the right ventricle was moderately dilated; the lung pathology was classic.

Congestive failure as a complication of bronchiolitis was emphasized by Henderson and Rosenzweig[4], who studied electrocardiograms on the 45 sickest infants with bronchiolitis in a series of 457. Of these 45, 37 per cent had abnormalities, the vast majority being ST displacement or abnormal T waves in Leads 1 and 2. These authors felt that cardiac pathology was due to hypoxemia, toxemia, and obstruction of pulmonary circulation, and that digitalization was important in management. Wood et al. and Nelson mention the occurrence of failure, and Wood suggests digoxin as treatment. Pratt had described the appearance of marked tachycardia with gallop rhythm, poor quality of heart sounds, and sudden hepatomegaly in severe bronchiolitis. In a study of bronchopneumonia in children Abt and Vinnecour[10] took serial electrocardiograms on eight children with no findings related to cardiac dysfunction, and found that during the acute illness all had small amplitude T-2 and many had variable changes in other complexes. They felt that the electrocardiographic abnormalities were due to toxemia and anoxia. In contrast to the above authors, Hubble and Osborn felt that the complicating problem in bronchiolitis is general circulatory collapse, and that cardiac glycosides have no place in treatment, the drug of choice being coramine. Although they described pulmonary pathology in this condition, they made no mention of cardiac pathology.

CONCLUSION

Considering our findings in the light of those of other authors, it would seem that acute cardiac failure is a very real, if rather infrequent, complication of bronchiolitis. Although obstructed pulmonary circulation suggests itself as a mechanism for failure, the universal lack of comment regarding the pulmonary vessels implies their normal appearance grossly and histologically. A more likely explanation would be damage to the myocardium from toxicity and anoxia. Several of the studies mentioned previously showed that the heart in bronchiolitis, pneumonia, and other infectious diseases may show abnormalities by electrocardiogram and histologic examination. Two of our 8 patients had definite conduction defects on electrocardiogram, and 1 of these showed right ventricular dilatation at autopsy. The dramatic response to digitalization in 7 of the 8 is indication that cardiac glycosides have a place in the management of cardiac failure complicating bronchiolitis.

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MECONIUM PERITONITIS

J. William Oberman, M.D.

Meconium peritonitis may be defined as a non-bacterial foreign body and chemical peritonitis occurring during intra-uterine life and caused by the escape of sterile meconium into the peritoneal cavity through a perforation in the bowel wall[1]. This definition excludes all neonatal peritonitis not associated with antepartum, intrapartum or shortly postpartum intestinal perforation; it also does not include any peritonitis resulting from perforation occurring after 3 to 6 hours following birth since it has been shown that the originally sterile intestinal contents become contaminated with bacteria after this time.

CASE REPORT

J. H., an 8 day old colored male, was admitted to Children's Hospital on December 14, 1954 with a history of vomiting blood-tinged fluid for 12 hours prior to admission. He was the product of a completely normal pregnancy and uncomplicated delivery. He did well in the hospital in which he was born and went home on the third hospital day. Birth weight was 6 pounds, 10 ounces. The neonatal hospital chart revealed no record of any meconium stool in the hospital. At home he was breast-fed and received supplementary formula. He ate well and seemed to thrive but vomited a portion of each feeding shortly after being fed. The vomiting gradually became more severe until the day of admission when it was almost continuous and had become blood-tinged. For the 3 days prior to admission he had been noted by his mother to have 1 to 2 small greenish bowel movements daily.

Physical examination on admission to the hospital revealed an acutely ill infant with cyanosis of the lips and nail beds. His temperature was 98°, pulse 130 and respirations 20. Auscultation of the lungs revealed bilateral rhonchi. The abdomen was soft, was not distended and was normal upon percussion. The liver was palpated

at the costal margin but no other organs or masses were palpable. No peristalsis was heard. An easily reducible hernia was present in the right inguinal region.

Initially the infant was placed in an incubator and given nothing by mouth; intravenous fluids were administered through a venous cutdown. In the hospital he was observed to be regurgitating blood-tinged fluid almost continuously. He had one soft dark-brown bowel movement shortly after admission. The child remained critically ill and began having generalized convulsions soon after admission. These convulsions were controlled with phenobarbital but he then began having short periods of apnea.

Initial blood and urine examinations were normal. X-ray of the abdomen (figure 1) revealed a large gas-filled stomach with very little air scattered through the remainder of the gastro-intestinal tract. There was a generalized frosted-glass density of the entire abdomen with multiple areas of calcified stippled material throughout

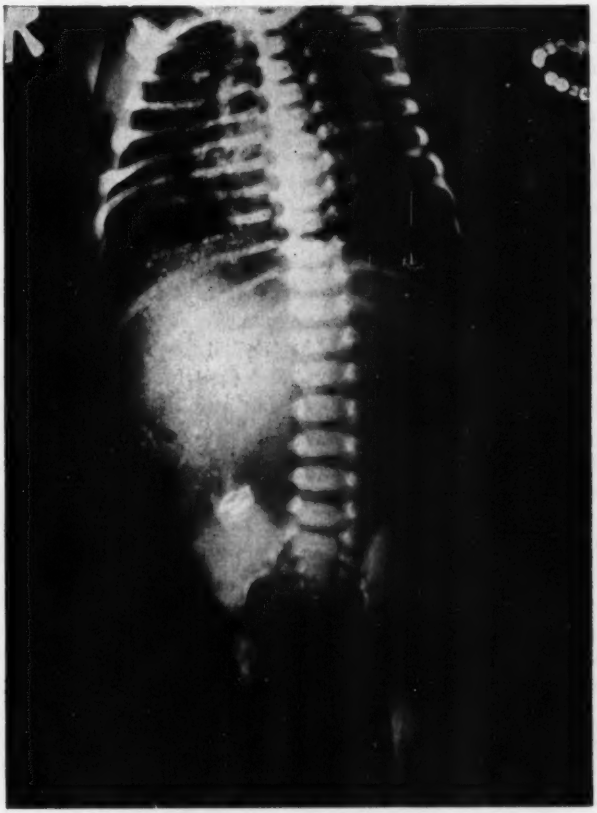

FIG. 1. X-ray Evidence of Meconium Peritonitis

the peritoneal cavity, more noticeable beneath the diaphragm, over the liver and on the right side of the abdomen and in the pelvis. These roentgenographic findings were interpreted as indicating fetal meconium peritonitis. X-ray of the chest revealed bilateral hilar infiltrations.

Following the convulsions, spinal tap revealed 113 leucocytes, 98 per cent of which were lymphocytes, a protein of 90 mg. per cent and a sugar of 250 mg. per cent. Spinal fluid culture was sterile. Tetracycline and chloramphenicol were administered intravenously. About 12 hours after admission, examination of the lungs revealed numerous coarse rhonchi; the heart rate was 160; and the liver was palpable 3 cm. below the costal margin. Because he was thought to be in congestive heart failure, the baby was given cedilanid intravenously without significant clinical response. He became febrile with a rectal temperature ranging from 99° to 101°. Despite all therapy he expired 30 hours after admission.

At autopsy the abdominal cavity contained greenish-yellow mucoid material spread over the entire peritoneum. There were multiple 0.1–0.2 cm. hard yellow nodules on all peritoneal surfaces especially over the diaphragm, liver and spleen, and pelvic organs. There was an annular pancreas associated with atresia of the duodenum. The duodenum was greatly dilated proximal to the obstruction but no perforation was found. There was a perforation of the hepatic flexure of the colon which was attached to the under surface of the liver by moderately firm adhesions. Examination of the lungs revealed a generalized severe necrotizing bronchopneumonia. Intermediate coliform bacilli were cultured from heart blood but culture of peritoneal material was sterile.

ETIOLOGY

Since meconium has been found in the intestine of a 3 month embryo[3] it would be theoretically possible for meconium peritonitis to occur at any time after the first trimester of pregnancy. However, since intestinal peristalsis, which is probably necessary to cause rupture of the bowel, has not been observed prior to the fifth month of pregnancy, it seems probable that meconium peritonitis can not occur before the last half of gestation[2]. In many instances the site of perforation can not be located and it is presumed that the perforation has had sufficient time to heal. Frequently, however, the perforation is readily located. There is usually only a single tear in the bowel wall although there are occasional reports of multiple perforations. These perforations vary from pinhead size to extensive tears involving half the circumference of the intestine.

It is estimated that 50 per cent of cases of meconium peritonitis are secondary to congenital lesions of the intestinal tract. In these cases, perforation of the intestine usually occurs just proximal to the site of the congenital lesion. These lesions give rise to intestinal obstruction which is soon followed by gangrene and resultant perforation[2][4]. Congenital gastro-intestinal lesions which have been implicated include malrotation of the bowel with adherent bands and/or volvulus, congenital (Meckel's) or acquired diverticula, intestinal stenosis or atresia, duplication of the bowel and strangulated inguinal hernia[4]. Meconium peritonitis may also follow intestinal obstruction due to meconium ileus.

Trauma is believed to play a role in many cases of meconium ileus developing either intrapartum or just postpartum. Perforations occurring at these times are said to be associated with trauma in almost 50 per cent of cases[5]. Factors involved probably include the pressure exerted during labor plus local intestinal distension with or without intestinal obstruction.

In the remaining half of instances of meconium peritonitis there is no evidence of any intestinal obstruction and in many instances neither the etiology nor the actual site of the perforation has been found. In cases without obstruction the sites of predilection of intestinal tears are the colon and terminal ileum. Suggested etiologies include abnormalities of the intestinal glands (deeply penetrating crypts of Lieberkühn), lymphatic tissue, or muscle layers (marked thinning of the circular muscular layer[2][3]. Some authors believe that most of the instances of meconium peritonitis unassociated with congenital gastrointestinal lesions are not *true* meconium peritonitis but occur after delivery[3]. The presence of meconium is believed to be only incidental to a pyogenic peritonitis.

CLINICAL MANIFESTATIONS

The diagnosis of meconium peritonitis is rarely made on the basis of clinical findings alone[1][6]. In those cases in which there is an associated congenital malformation of the intestinal tract, the symptoms and signs become those of intestinal obstruction, i.e., progressively increasing vomiting, abdominal distension and failure to pass a stool. Meconium peritonitis may cause death in utero and a stillbirth results. Many cases of meconium peritonitis unassociated with intestinal obstruction heal in utero and are never diagnosed. Death or abdominal operation for an entirely unrelated condition in these instances reveals nodules of calcified meconium accompanied by dense bands and adhesions. A more striking symptom complex is presented by those infants in whom perforation occurs just prior to, during, or immediately following delivery. In these the most prominent sign is abdominal distension, often so marked as to interfere with normal delivery[4]. These infants are usually born with grunting respirations and cyanosis and have a universally poor prognosis.

X-RAY FINDINGS

Neuhauser[1] first described the roentgenographic findings which, if present, are considered to be pathognomonic of meconium peritonitis. X-ray of the abdomen (usually taken because intestinal obstruction is suspected) reveal many irregular shadows of calcific density scattered throughout the peritoneal cavity and lying outside the dilated loops of bowel, within the bowel lumen or loosely adherent to the peritoneum. These areas represent nodules of calcified meconium. Calcification of the meconium is said to

begin as early as 24 hours after the onset of peritonitis[2] so that this finding is not an absolute differentiating point in the decision as to whether intestinal perforation occurred in utero or following delivery. Calcification may also be so slight as to be demonstrable only when x-rays are taken of tissue obtained at autopsy. X-ray of the abdomen in those cases in which perforation occurs in close relation to the time of delivery frequently reveals a pneumoperitoneum. However, both pneumoperitoneum and peritoneal calcification occur in only a certain percentage of cases. In addition, calcified meconium occasionally occurs within the lumen of an intact gut and in the absence of peritonitis[3].

PATHOLOGY

Boikan[2] states that the following criteria need to be met in establishing the diagnosis of meconium peritonitis at autopsy: the presence in the peritoneal cavity of meconium, calcified meconium, mucus droplets, foreign body giant cells, cells from the source of perforation, fibrinous or fibrous adhesions and rarely, lanugo hair. The calcified meconium is represented by many pinhead to pea-sized yellowish lesions scattered through the peritoneal cavity and lightly adherent to the omentum. There may be larger patches having scattered calcareous nodules and areas of fat necrosis[3]. Usually the perforation of the intestine has closed and cannot be identified. Close search will frequently uncover an anomaly of the intestinal tract.

If perforation should occur a short time before birth, in the abdomen, recent adhesions, early calcification, and possibly recently extruded meconium are all found at autopsy.

PROGNOSIS

Because many cases of meconium peritonitis are undetected, reliable information as to true incidence or prognosis is lacking. Undeniably many infants remain completely asymptomatic and live normal healthy lives. Others are born dead. Of those born alive in whom symptoms arise, Toio and his co-workers[4] in 1954 collected information on 15 cases of meconium peritonitis who were successfully operated on and survived. The time of operation varied from 2½ hours to 4 days after birth and the type of operation varied from suture of the perforation and simple drainage of the abdomen to single or multiple resections with one-stage or multiple stage anastomoses. Only 5 of these 15 survivals had abdominal distension at or within a few hours after delivery, emphasizing the relatively poor prognosis of this group of recent perforations. Bacterial contamination occurs rapidly in these cases and a fatal outcome is the rule.

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INTUSSUSCEPTION

Case Review

Robert L. Bregman, M.D.

Intussusception is the most common cause of acquired intestinal obstruction in infants and children. It is a condition that is likely to be mimicked by many diseases seen every day in pediatric practice.

Judicious use of the barium enema, improved pre- and post- operative care, with special attention to electrolyte balance, and good surgical technique have resulted in markedly reduced mortality from intussusception in recent years. Despite such advances, children still die with intussusception and the mortality is directly proportional to the time interval from the onset of symptoms until the institution of proper therapy.

Careful evaluation is essential for the infant whose mother states that her baby is "fussy", "colicky", or has "cramps". Proper attention to such vague symptoms and consequent early diagnosis of intussusception may spare the infant an unnecessary and difficult intestinal resection.

A reliable history is of prime importance in establishing an early diagnosis of intussusception as the patient may seem perfectly well if examined early or between paroxysms of pain. The physician who is alert to the possibility of intussusception can then forewarn the child's parents of other signs and symptoms which may develop. If this condition is diagnosed early, a procedure as simple as the barium enema may result in a complete cure whereas delay may necessitate intestinal resection in a critically ill child.

The medical records of 45 patients with proven intussusception seen in Children's Hospital over a 5 year period have been reviewed. An attempt has been made to correlate the information from these cases with previously reported series and to evaluate different methods of therapy. Seven additional cases with a typical history and physical findings of

intussusception which regressed spontaneously have been observed over the same period of time.

DEFINITION

Intussusception is defined as the telescoping of a portion of small intestine or colon into a more distal segment of the enteric tube. If proper therapy is not instituted the process is almost invariably a progressive one eventually resulting in the death of the child.

ETIOLOGY

In the majority of instances of intussusception, (90–95 per cent), no etiological factor can be found. In our 45 cases there were 14 in which a particular etiology was suggested. Mesenteric lymphadenopathy was reported as a likely etiological factor in 9 cases and Meckel's diverticulum in 3 cases. Among the more unusual causes were a vascular band at the base of the cecum and a band of fatty tissue across the ileocecal junction.

It is well known that intussusception may occur during or after an episode of acute gastroenteritis; possibly due to an alteration in intestinal peristalsis. Seven patients in this series had diarrhea for several days prior to the diagnosis of intussusception. Constipation often precedes the development of intussusception and was noted in 3 patients in our series.

Nelson lists the following other conditions as possible predisposing factors in the development of intussusception: drastic purgation, colic, abdominal injuries, ulceration and hemorrhage in the intestine, foreign bodies, large fecal masses, parasites, polyps, cysts and tumors.

LOCATION

In our series the most common type listed was the ileo-colic variety. Others were ileo-cecal, ileo-ileal and colo-colic. There were 3 cases with double intussusception, ileo-cecal and colo-colic, ileo-ileal and ileo-cecal, and ileo-ileal with ileo-colic. There appears to be little or no clinical significance attached to the various types of intussusception.

SEX AND AGE DISTRIBUTION

In this series 71 per cent of the cases occurred in males. In a larger series, approximately 65 per cent of the cases were boys. The patients are usually well nourished and above average in physical development. This very healthy appearance may be deceptive and lead to a mistaken diagnosis. This was graphically illustrated in one of our cases who was sent home with the diagnosis of a behavior problem and overanxious parents and who returned several hours later with the classical picture of intussusception.

Our patients ranged from 2½ months to 9 years of age. Fifty per cent

of these children were under 1 year of age and 75 per cent were under 2 years. The youngest patient reported by Gross was 3 days old and 75 per cent of his cases were under 1 year. The change from liquid to solid foods in infancy has been suggested as a factor in the age distribution, however, there is no apparent reason for the predominance of males.

SIGNS AND SYMPTOMS

The leading symptoms in our series included vomiting, the passage of bright red blood in stools and apparent abdominal pain. These occurred in 75–85 per cent of the cases as the chief complaint.

The onset of intussusception is usually sudden and alarming with recurrent colicky abdominal pain occurring in a child who has been previously well. The child may cry or grunt as if in pain and flex the thighs on the abdomen. This occurs in paroxysms, lasting 10–15 seconds, following which the child becomes relaxed, seems well and may play normally, only to have recurrent pain 10–20 minutes later. Repeated and severe vomiting usually quickly follows the onset of pain. Bright red blood occurs in the stools 12–24 hours after the onset of symptoms. The blood is most always mixed with mucus and is termed a "currant jelly" stool. The amount of bleeding varies and may be severe.

Other symptoms in our series were lethargy, irritability, loose stools, constipation and anorexia. If untreated the patient becomes dehydrated quickly and frequently becomes moribund in 2 or 3 days. The duration of symptoms in our series varied from 2 hours to 3 weeks prior to hospitalization. Fifty per cent of the patients were hospitalized within 24 hours and another 1 per cent within 48 hours after onset of symptoms.

The physical findings vary according to the duration of symptoms and the time relation between examination and the paroxysms of pain. Early examination may be completely normal. Later the child appears acutely ill, dehydrated and pale. He may have a rapid pulse, low grade fever and often cries and thrashes about periodically. In this series a mass was palpated in only 50 per cent of the patients. In other series the mass is said to be palpable in 85–90 per cent of the cases. It is usually described as sausage shaped, firm and non-tender; however, the baby often winces with palpation of the abdomen. The mass may be palpated in any location in the abdomen and may occasionally be felt by rectal examination. This examination should be performed routinely in all suspected cases and usually discloses evidence of dark blood.

In 30 per cent of our cases the temperature was 100–101° rectally. In the remaining cases it was normal with one exception. This patient had a four day history and a temperature of 105° on admission. The intussusception was easily reduced surgically without resection, however, the patient

expired several hours post-operatively. Autopsy revealed necrosis of the ileum with peritonitis. This was the only fatal case in this series; a temperature over 101° is considered to be a poor prognostic sign.

Laboratory studies are of little or no diagnostic value. The urinalysis usually reflects the state of hydration and acidosis. In 33 per cent of our patients there was a leucocytosis over 15,000 with a shift to the left. The remainder had normal blood counts.

DIAGNOSIS

The diagnosis of intussusception is based on the typical history and physical findings previously described. In many cases X-ray of the abdomen will show evidence of small bowel obstruction. Gross states that approximately 5 per cent of cases require barium studies for diagnosis. In our series only one patient required this procedure. This was a 3 year old girl with an 11 day history of intermittent abdominal pain. Physical examination was negative and there were no other symptoms. Barium enema revealed an ileo-cecal intussusception. Exploration confirmed the diagnosis and a vascular band around the base of the cecum was found and thought to be the causative factor.

Barium enema in a patient with intussusception reveals the following: 1) obstruction to the retrograde injection of barium; 2) cupping in the head of the barium column as it meets the intussusceptum; 3) a thin casing of barium surrounding the intussusceptum; 4) a cylindrical shell of barium surrounding and outlining the intussusceptum, which remains after evacuation of the bowel; and 5) gaseous distention of the ileum just proximal to the intussusception. The intussusception may be partially or completely reduced by the barium.

The differential diagnostic possibilities are numerous. Simple colic, gastrointestinal allergy, non-thrombocytopenic purpura and a bleeding Meckel's diverticulum may all simulate intussusception. Acute appendicitis in an infant may be difficult to differentiate and intussusception must be ruled out in any atypical case of dysentery. Non-specific diarrhea and acute appendicitis were the most frequent erroneous admission diagnoses in this series. One patient was diagnosed as having conversion hysteria and another as having a blood dyscrasia on admission.

TREATMENT

There are two methods of treating intussusception: surgical reduction and non-operative radiologically controlled hydrostatic pressure reduction. The results, in capable hands, by either method are very good and the surgical mortality is approaching the irreducible minimum.

Gross objects to the routine use of the barium enema for the following reasons: 1) it may delay operation if there is only partial reduction or if the physician is doubtful whether or not reduction has occurred; 2) colonic inflation has limited effectiveness above the ileo-cecal valve; and finally 3) an ileal polyp or Meckel's diverticulum will be completely overlooked during barium reduction. He suggests universal surgical treatment if proper facilities and personnel are available.

Ravitch and McCune, on the other hand, suggest that all patients be treated by radiologically controlled hydrostatic pressure reduction, followed by operation if necessary to confirm reduction, or to complete the reduction if the intussusception persists. They recommend that all children undergo this type of therapy regardless of the duration of symptoms. The non-operative management has the advantage of much less discomfort for the patient and a shorter period of hospitalization; however, recurrences are more likely to occur after hydrostatic pressure reduction. There were no cases of recurrent intussusception in this series.

Sixteen of the 45 cases were treated by attempted hydrostatic pressure reduction. Six of these were completely reduced whereas the remaining 10 cases were incompletely reduced or not reduced at all. In none of these 10 cases were symptoms present for more than 48 hours.

The remaining 29 cases were treated by surgical reduction initially. Twenty-seven were simple reductions and 2 cases required intestinal resection. All of the patients made an uneventful recovery with the exception of the one fatality previously described. An exploratory laparotomy was performed on one other patient on the eighth postoperative day because of obstruction. This patient had developed a volvulus of the ileum which was reduced without difficulty. Following this he made an uneventful recovery.

An appendectomy was performed in 11 of the 29 cases because of hemorrhage or infarction in an appendix which had been caught in the intussusception. As a general rule during the operative reduction of an intussusception elective surgery should be deferred. Any procedure which would open the intestinal tract in a previously damaged bowel would naturally increase the possibility of peritonitis, however; if the patient is in excellent condition, if his symptoms are of brief duration, and if the intussusception is easily reduced, appendectomy may be done without fear of further complications.

The decision to resect intestine must be made during surgery after careful evaluation of the viability of the bowel. With early diagnosis and treatment, the necessity for resection will be virtually eliminated.

In summarizing the treatment of intussusception there can be little

argument that the majority of patients will do well regardless of the method of therapy provided that this therapy is instituted as soon as possible after diagnosis. The patient who has had symptoms for 12–24 hours will probably be adequately hydrated and in good condition on admission to the hospital. It would appear that this patient should have an attempt at radiologically controlled hydrostatic pressure reduction. Naturally, surgery would follow immediately if there was any doubt as to completeness of reduction. The operating team should be alerted upon admission of the patient to the hospital.

If the patient is dehydrated and acidotic as many will be, regardless of the duration of symptoms, the immediate treatment should be restoration of fluid and electrolyte balance and transfusions if indicated. While being rehydrated the patient may undergo hydrostatic pressure reduction if the symptoms have not existed for more than 24 hours.

If symptoms have existed for more than 24 hours, surgery appears to be the treatment of choice after corrective fluid therapy. The 6 cases in this series successfully reduced by barium were of less than 24 hours duration. One of the 10 cases not reduced was of 16 hours duration and the remaining 9 were from 24 to 48 hours duration. It is unwise to perform immediate surgery in the critically ill patient. The delay of several hours, during which total fluids and electrolytes will be restored to normal, may enable the patient to survive an extensive surgical procedure which may have been fatal in the severely dehydrated state.

If the hydrostatic pressure reduction is used it is recommended that the barium be held 3 to 3½ feet above the fluoroscopic table and that reduction be accomplished by gravity above and not by manual manipulation of the abdomen. If this method is used, rupture of the bowel will not occur nor will gangrenous bowel be reduced. No more than 3 attempts at barium reduction are justified.

For successful treatment the barium should be seen entering well into the small intestine and should be returned with flatus or feces. The mass should disappear and the patient should show clinical improvement. Charcoal should be given by mouth and subsequently be recovered in a blood free stool.

It is strongly recommended that the surgeon who will be responsible for the decision to operate be in attendance during the hydrostatic pressure reduction. Unfortunately, too often this procedure is carried out by the pediatrician and radiologist and later the surgeon is called without having had the benefit of observing the fluoroscopic examination.

Patients with recurrent intussusception should be treated surgically, as an etiological factor is more likely to be discovered in such cases. There was a 2 per cent incidence of recurrences in Gross' series.

SUBACUTE AND CHRONIC INTUSSUSCEPTION

Subacute and chronic intussusception may occur with symptoms lasting 5 days to 3 or 4 weeks. These patients have partial intestinal obstruction and damage to the intestine and mesentery is minimal. They usually have a history of irregular vomiting and abdominal pain and intermittent bloody stools.

Barium enema is essential for diagnosis and prognosis is excellent in such cases. There were 3 cases in our series with subacute or chronic intussusception; all of them recovered uneventfully following surgery. Two of these patients had an ileo-ileal intussusception and the third patient had an ileo-cecal type.

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THE PROBLEM OF AVAILABILITY OF IMMUNE GAMMA GLOBULIN THROUGH HEALTH DEPARTMENTS

John R. Pate, M.D., M.P.H.*

The allocation of gamma globulin for the calendar years 1953 and 1954 has been administered by the United States Public Health Service on authority of the Office of Defense Mobilization. The American Red Cross participated in this program by turning over all of its gamma globulin to the Office of Defense Mobilization for allocation.

The Red Cross obtained its gamma globulin from fractionation of plasma of outdated or otherwise unusable blood obtained from its Regional Blood Program and from the Department of Defense. In the past two years the Red Cross has made available 10,354,638 cc. of gamma globulin for allocation to states and territories.

The National Foundation for Infantile Paralysis last year set aside several millions of dollars to help finance the production and distribution of gamma globulin for short-term, passive immunization of paralytic

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poliomyelitis. This was done in the hope that it would be of benefit while the field tests of poliomyelitis vaccine were being made on a nation-wide sampling. When the funds were exhausted, the Red Cross continued to provide gamma globulin, though of course in a drastically reduced quantity.

On October 1, 1954 gamma globulin was made available through commercial channels. On December 31, 1954 the Office of Defense Mobilization discontinued allocation of gamma globulin.

The only gamma globulin now available to the Red Cross is that obtained by processing unusable blood in its civilian program. Realizing that this amount is not sufficient to cover the needs of the entire country the Red Cross medical staff formulated a plan in February. It was determined that the distribution of the bulk of the Red Cross gamma globulin should be the responsibility of the state and territorial health officers in order to assure equitable and accessible distribution.

The allotment of Red Cross gamma globulin for Washington, D. C. during 1955 is estimated to be 8,610 cc. The delivery of allotments are to be made quarterly. At the present time the supply is packaged in 10 cc. vials since these were on hand but production of 2 cc. vials will be started. After the supply of the old 10 cc. packaged vials has been consumed, all new production will be in 2 cc. amounts.

The Medical Director of the American National Red Cross states, "We are aware that the demand will exceed the supply. Therefore, it will be necessary to refuse any requests over that which will be shipped to you in accordance with the schedule. A small quantity of gamma globulin has been reserved for research, investigational work, and individual cases requiring larger quantities. If your supply is insufficient to care for these individual cases, they may be referred to us." This directive has made it mandatory that local Public Health departments and Medical Societies set more rigid restrictions on distribution of Red Cross gamma globulin.

The allotment of gamma globulin by the Red Cross to an area is based upon the needs for it as determined by the accumulated incidence over the past several years of the diseases that benefit from gamma globulin. Therefore, it is of considerable importance that all diseases wherein gamma globulin is effective should be reported to the Department of Public Health.

As far as the District of Columbia is concerned, the Department of Public Health will make every effort to be as liberal as practical in the distribution of the product. The cooperation of the medical profession is required in supplying necessary information for record keeping purposes when requesting gamma globulin. The department will be pleased to participate in consultation with any physician relative to a problem concerning gamma globulin.

EDITORIAL

CURRENT AVAILABILITY OF IMMUNE GAMMA GLOBULIN THROUGH HEALTH DEPARTMENTS

EDITORIAL SERVICE

On October 1, 1954 pooled gamma globulin was made available through commercial channels and on December 31st the Office of Defense Mobilization discontinued the allocation of gamma globulin. This meant that supplies of globulin available through the Red Cross and the state and territorial health officers would be considerably limited as compared to recent years. This fact in turn has necessitated closer supervision of the distribution of immune gamma globulin by county and district health departments, as pointed out by Dr. John R. Pate in this issue. For this reason, *Clinical Proceedings* has canvassed the Health Officers of the District of Columbia and the surrounding counties for information concerning the current availability of immune gamma globulin through their departments. A summary is presented here in table form for quick reference. There is included a summary of possible indications and recommended dosage based on the replies of these Health Officers.

Please turn to the next page for the table of recommended use and dosage of prophylactic gamma globulin showing the availability of the product through health departments in the metropolitan District of Columbia area.

CHART 1

Recommended Use and Dosage of Prophylactic Gamma Globulin Showing Availability Through Health Departments In The Metropolitan District Of Columbia Area

Use	Recommended Dosage According to Days After Exposure	Availability					
		District of Columbia	Montgomery County	Prince Georges	Arlington	Alexandria	Fairfax
Measles							
Prevention	0.1cc/lb under 6 days	Up to 3 years	Up to 3 years	Up to 3 years	Up to 2 years	Up to 3 years	Up to 2 years
Modification	0.02cc/lb under, 0.1 cc/lb over 6 days	On consultation	3 to 6 years	Up to 3 years	2 to 5 years	3 to 5 years	2 to 5 years
Institutions	On consultation	On consultation	On consultation	On consultation	On consultation	On consultation	On consultation
Infectious hepatitis							
Prevention, household contacts	0.01–0.02 cc/lb under 7 days	On consultation	+	+	Up to 2cc	+	+
Prevention, schools, institutions		On consultation	+	On consultation	On consultation	On consultation	On consultation
Rubella							
Prevention, pregnant woman, first 4–5 months	0.1cc/lb ? Effectiveness	On consultation	+	+	+	+	+
Poliomyelitis							
Prevention	0.2cc/lb up to 10cc under 5 days	Not recommended	On consultation	Under study	On consultation	Under study	Under study
Agamma-globulinemia	Varies individually	Contact Red Cross Area Director	On consultation	On consultation	On consultation	On consultation	On consultation
Name of responsible health official		John R. Pate, M.D.	Wm. J. Peeples, M.D.	T. S. Englar, M.D.	R. G. Beachley, M.D.	T. F. McGough, M.D.	Harold Kennedy, M.D.

CORRESPONDENCE

"PROTHROMBIN STUDIES IN PEDIATRICS"

The following exchange of letters concerns an article published in the July, 1954, Clinical Proceedings.

February 22, 1954

Editors:

In the July, 1954, *Clinical Proceedings of Children's Hospital* there appeared a paper entitled, "Prothrombin Studies in Pediatrics" by Dr. Jean Lockhart, which attempts to evaluate the blood prothrombin test with currently available Quick whole plasma and diluted plasma prothrombin determinations.

"In an effort to determine the practicality and accuracy of a capillary blood prothrombin test", reference is made by the author to "Dr. Benjamin Manchester's recent exhibit" at the District of Columbia Medical Society Convention. The above procedure was described to Dr. Lockhart for several days. The method of preparation of thromboplastin, the technique for performing the test was demonstrated and repeatedly supervised, in order to be certain that the results would be reduplicated. Emphasis for the storage, care of the glassware, and technique for warming the slides as well as thromboplastin when in use, were repeatedly advised.

The accuracy of this test is dependent upon an active thromboplastin. Because the reagent is an enzyme, it is, therefore, unpredictable in its activity. It was emphasized (to Dr. Lockhart) that a blood prothrombin determination must always be made on a control prior to its use on the test individual. The control must yield a 14 to 18 seconds with a standard deviation of 2.5 seconds and if the prothrombin time is in excess of 21 seconds, the thromboplastin was inactive due to aging, adulteration, or due to the fact that it had not been warmed properly and, therefore, remained inactive.

It is apparent from Figure 2, page 129, that these simple precautions were not taken seriously in 14 of the 20 tests that are recorded. In only six, were the control prothrombin determinations within the range of normal. The author presumes to estimate prothrombin activity without regard for this simple precaution.

The writer is disturbed that conclusions were drawn, not validated by any of the data recorded. Little mention is made of any references to our own publications, 1, 2, 3.

The present criticism is made in order to emphasize, as we have in the past, that our simple blood prothrombin test is no more accurate than the individual who performs it. Dr. Lockhart's report confirms the validity of this observation.

I regret that such a letter is necessary.

Yours very truly,
Benjamin Manchester, M.D.

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Editors:

Thank you for your letter of March 7th, enclosing a copy of a communication from Dr. Benjamin Manchester regarding my study of Prothrombin Studies. (*Clinical Proceedings*, July, 1954)

At the time I made the prothrombin determinations on newborns at Garfield, I felt more keenly than ever the need for a reliable capillary test, since in many instances I was unable to draw 5 cc. of venous blood from the baby fast enough so that it did not clot! I had hoped to test fifty or more babies, and at the end of two months had completed only 20 tests, some of those on the same baby on successive days. Therefore it was with more than ordinary enthusiasm that I followed the directions outlined to me by Dr. Manchester and his competent technician, in the performance of the capillary prothrombin test devised by Dr. Manchester.

I would like to say again, as in my paper, that *neither* the venous nor the capillary prothrombin times proved to be very consistent, with expected values in newborns or with day by day results. The venous blood tests were performed by the Garfield Laboratory, and I tested their results by sending them a prothrombin test of my own venous blood (labeled Baby L), with the result that I had 100 per cent activity. In other words, even the accurately performed Quick method did not produce very consistent results on these babies.

Dr. Manchester is perfectly correct in his criticism of my use of thromboplastin which did not yield a control under 18 seconds. The technical problem involved was this: after mixing and incubating of the dry thromboplastin, according to his directions, it was stored in the deep freeze. Because of the limited number of tests I performed each day, I so prepared only one tube of thromboplastin at a time. Not infrequently it happened that, despite apparently adequate heating of the thromboplastin, testing of the control on the first day of use of that batch of prothrombin did not time under 18 seconds. In retrospect, I should have prepared several batches of thromboplastin, and heated each until one met the criteria established by Dr. Manchester; however, what I actually did was to use the thromboplastin on hand and record the findings, and then discard that tube of thromboplastin.

There is a great need for a reevaluation of capillary prothrombin tests, and the very limited studies performed by me were in no way intended to settle the question as to the merits of any one test. I do feel that no amount of theory is any better than the technical results of a test, and that Dr. Manchester's very excellent capillary prothrombin test deserves more study with more refined technique.

Sincerely,
Jean D. Lockhart, M.D.

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